CND1 G870A and *XPD* Lys751Gln variants were significantly presented in cases as compared to controls (age adjusted OR %CI 1.7-3.4) and 1.7 (95%CI 1.3-2.4) respectively. A significant ene interaction was also observed among cases with two variant for both *CCND1* and *XPD* genes (OR 8.0, 95%CI 2.6-24.2). rs with at least one *CCND1* variant allele had an OR of 3.7 . 2.1-6.3) as compared to non smokers, OR 1.3 (95%CI 0.7-2.4). ly smokers with at least one *XPD* Lys751Gln variant allele had an 3.0 (95%CI 1.7-5.6) as compared to non smokers, OR 1.07 l 0.6-1.9). Moreover, smokers with a combination of at least one allele of both *CCND1* and *XPD* genes have an OR of 17.0 I 7.4-39.4) as compared to non smokers, OR 6.3 (2.6-15.0). results suggest an interaction between polymorphisms in genes nucleotide excision repair pathway, cell cycle control pathway, ure to carcinogens present in cigarette smoke and upper aero- ve tract cancer risk. [Supported by NIH 5U01 CA084968]

178 **The role of insurance and hospital in the short term al of young hispanic and caucasian women with advanced al cancer in southern California.** Alessandra Re, Argyrios Zio- oda Anton-Culver, and Wendy Brewster, *University of California Medical Center, Orange, CA, University of California Irvine, Irvine,*

ious studies have demonstrated differential cervical cancer sur- etween women of differing ethnic groups in the United States. I describe the short-term outcome of non-Hispanic- White (NH- and Hispanic women with advanced cervical Cancer (stage 3) in Southern California with respect to insurance status, treating al and income. Women less than 55 years at the time of diagnosis anced cervical cancer diagnosed between 1994-1999 in Orange an Diego Counties, California were identified. Subjects were ed based on: type of insurance -None/Unknown, State/Federal rivate; type of hospital -Teaching/Academic, Community and One hundred thirty four subjects were identified (59 Hispanic and White). Mean disease specific survival was 32.1 months versus nonths in favor of Hispanic women (p =0.31). There was no cal significant difference in the delay to treatment based on nce or hospital stratification between the two groups. The more ed the stage of disease the longer was the delay; stage IIB - 29.5 stage III - 33 days and stage IV - 47 days. There was a difference ase specific survival at 18 months in favor of Hispanic women 79, 95% confidence interval 1.03 - 3.14). This appeared limited to ic women with private insurance 60.3% versus 87.1% (p=.04). tivariable analysis predictors of survival were race, income, in- e type, and stage. Ethnicity and income are significant factors in tcome of young women advanced cervical cancer with Hispanic n having a survival advantage over NH-White women. Insurance is a less significant contributor to short-term outcome. The poor term outcome in the group of NH-White women deserves further gation.

179 **Heterozygosity for hemochromatosis is associated ung but not head and neck cancers.** Juan Rodriguez-Paris, n Smith, Lisa Requena, Rose Marie Robertson, Glenn Mills, Jerry ty, Jonathan Glass. *Feist-Weiller Cancer Center, Louisiana State sity Health Sciences Center, Shreveport, LA.*

editary hemochromatosis (HH) is a common, autosomal recessive fer of iron metabolism caused by a mutation in the HFE gene on osome 6. The mutation causes an increase in iron absorption, results in excess deposition of iron in the parenchymal cells of le organs resulting in diabetes, liver failure, cardiac dysfunction, is, and hypogonadism. In the United States approximately 10% Caucasian population are carriers for the disease. HH heterozy- have slightly increased iron absorption, which may result in sed total body iron. Recent studies suggest that HH heterozy- may have an increased risk for developing cancer and heart se. As the generation of active oxygen species is implicated as l in lung cancer and head neck cancers and as iron catalyzes the ation of active oxygen species, we did a case-control study to mine if HH heterozygotes carry an increased risk for either cancer More than 90% of hereditary hemochromatosis patients have a missense mutation, causing an amino acid substitution of cys- to tyrosine at residue 282 (C282Y) in the HFE protein. We screened for the C282Y mutation in 176 Caucasian patients diagnosed with lung (87) or head and neck (83) cancer and 391 matched controls. All the patients were smokers. Genomic DNA was isolated from whole blood and the C282Y mutation was detected by allele-specific PCR. Of the 176 cancer patients 25, or 14.2%, were heterozygotes compared to only 10.0% of the 391 controls. The prevalence of the C282Y mutation was significantly higher among female patients, 17.7% (p=0.01), than in female controls, 9.6%. Adjusted for age, gender family history of cancer and smoking, females with the C282Y mutation were more likely to have lung cancer than female with no mutation (adjusted OR=6.8, 95% CI 1.33 to 34.6) and showed an earlier age of onset of lung cancer than female patients with wild type HFE, 51.2 years vs. 57.1 years respectively. Interestingly, the family history of cancer in first-degree relatives of HH heterozygotes appeared to be increased. The increased risk of cancer was present only in female heterozygotes with lung cancer and was not present in females with head and neck cancer or males with either cancer type. The causes of the significantly increased risk of lung cancer with the heterozygous state among females are being investigated.

#LB-180 Reproductive factors and risk of glioma in women. K. Huang, E.A.Whelan, A.M.Ruder, E.M.Ward, J. Deddens, K.E. Davis-King, T. Carreón, M.A. Waters, M.A. Butler, G.M. Calvert, P. Schulte, Z. Zivkovich, E. Heineman, J. Mandel, R. Morton, D. Reding, K. Rosenman, *and the Brain Cancer Collaborative Study Group, National Institute for Occupational Safety and Health, Cincinnati, OH.*

Reproductive hormones may play a role in the development of glioma in women, but few studies have examined this association. As part of a population-based case control study, histologically confirmed primary glioma cases (n=341 women) diagnosed between 1995 and 1997 were identified through clinics and hospitals in four Midwest U.S. states. Controls (n=528 women) were randomly selected from lists of licensed drivers and Health Care Finance Administration enrollees. In-person interviews with subjects (81%) or their proxies (19%) collected reproductive history and other exposure information. Glioma risk increased with older age at menarche (p for trend = 0.0076), but only among post-menopausal women. Among pre-menopausal women, those who had ever experienced a live birth were over two times as likely to be diagnosed with glioma than were nulliparous women (odds ratio (OR)=2.7, 95% confidence interval (95% CI)=1.3-5.3). Compared with women who breastfed 1-3 months over their lifetime, women who breastfed 4-8, 9-18 or 18 months were at increased risk (OR=1.6, 95% CI=0.7-3.9; OR=3.0, 95% CI=1.4-6.5; OR=4.6, 95% CI=2.1-10.2; respectively) (p for trend=0.0008). Women who used hormones for symptoms of menopause had a significantly decreased risk of glioma compared with women who never used such hormones (OR=0.6; 95% CI=0.4-0.9). A high cumulative number of menstruation months (438) was also associated with a reduced risk of glioma (OR=0.6, 95% CI=0.3-1.3). Our results support the hypothesis that reproductive hormones play a role in the etiology of glioma among women, with a pattern of associations that is almost the inverse of that observed for breast cancer.

#LB-181 A polymorphism in the FGF3 promoter and it's association with risk of esophageal squamous cell carcinoma. Yongjun Guo, Meghan McLaine, David Boorman, Dongxin Lin and Thomas G. O'Brien, *Lankenau Institute for Medical Research, Wynnewood, PA, and Cancer Institute, Chinese Academy of Medical Sciences, Beijing, China.*

FGF3 (fibroblast growth factor 3), originally named int-2, was first identified as an oncogene in mouse mammary tumors. It has been implicated in several types of cancer, including esophageal squamous cell carcinoma(ESCC), breast, lung, and head and neck carcinomas. By direct sequencing, we found a C/T single nucleotide polymorphism (SNP) in the 5'-proximal region of the FGF3 promoter. The polymorphism is predicted to create new transcription factor binding sequences in the FGF3 promoter. We therefore hypothesized that this SNP could have functional significance. We examined the association between this polymorphism and risk of esophageal squamous cell carcinoma in 168 ESCC cases and 171 age-, sex- and smoking status-matched controls from northern China. Genotyping for FGF3 was performed using a TaqMan assay developed in our laboratory. Results of statistical analysis show that compared to individuals homozygous for T-allele, those homozygous for C-allele had a more than 2-fold in-

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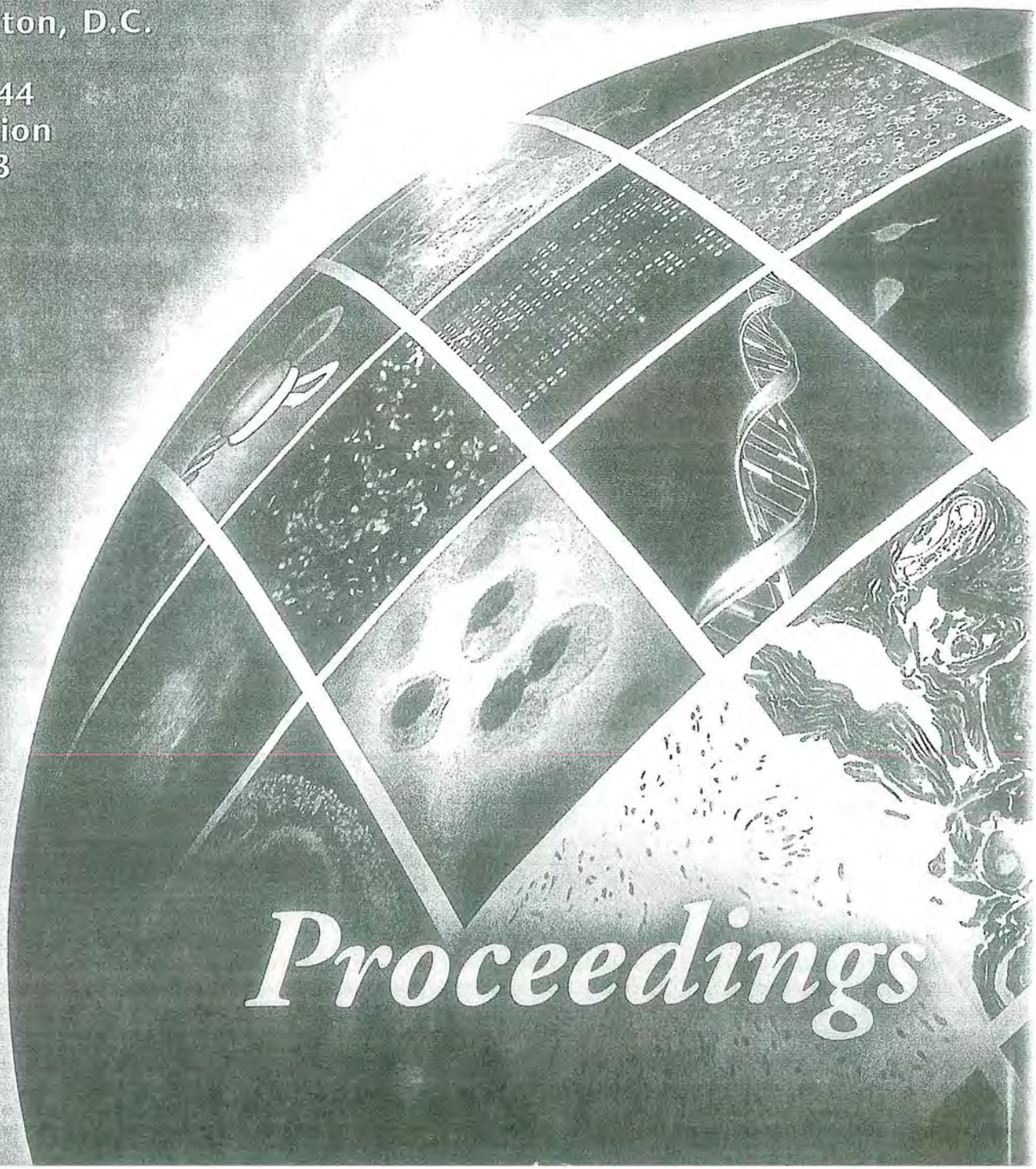

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